ENCLOSURE 4

I, STEVEN P. BRADBURY, declare as follows:

1. My name is Steven P. Bradbury. I hold a Doctor of Philosophy with a double major in Toxicology and Entomology (Insecticide Toxicology). I received this degree from Iowa State University in 1985. I also received a Master of Science degree in Entomology (Insecticide Toxicology) from the same institution in 1981. From October 1985 until September 1991 I held the position of Research Biologist (Toxicologist) in the Predictive Toxicology Branch at the Environmental Research Laboratory-Duluth, in Duluth, Minnesota. This facility is a U.S. Environmental Protection Agency laboratory within the Office of Research and Development. Since September 1991 I have been the Chief of the Predictive Toxicology Branch at the Environmental Research Laboratory-Duluth. I am an expert in the field of predictive and environmental toxicology, with an emphasis on industrial chemicals and insecticides.

2. I also presently hold a position as an adjunct assistant professor in the Graduate Faculty of Toxicology at the University of Minnesota. I have served as a reviewer for scientific journals in the field of environmental toxicology (including Environmental Toxicology and Chemistry) and have both chaired and been invited to attend numerous national and international conferences in this area of scientific study. A copy of my Curriculum Vitae is attached hereto as Exhibit A and is incorporated herein by reference.

3. My research efforts both in graduate school and as a professional have involved laboratory studies on the effects of industrial chemicals and insecticides on aquatic organisms and wildlife. I also direct the development of computer models, databases, and programs that assist regulatory staff to prepare assessments of the hazards of pollutants to aquatic life and wildlife. Overall, my duty as a research toxicologist is to estimate concentrations of pollutants in the nation's surface waters that, if not exceeded, can be reasonably assumed to protect aquatic organisms and wildlife that use freshwater as a source of food and drinking water.

4. As part of my professional activities, I have been collaborating with other U.S. EPA staff, as well as scientists from the State of Wisconsin Department of Natural Resources and the U.S. Fish and Wildlife Service to develop approaches for estimating concentrations of pollutants in surface waters that can protect wildlife. A major area of current collaboration has involved deriving criteria for the protection of wildlife in the Great Lakes Basin. It is intended that these criteria will be issued as guidance to States in the Great Lakes Basin pursuant to Section 118 of the Clean Water Act, 33 U.S.C. 1268. As part of our collaborative efforts, I have been involved in developing a water concentration of 2,3,7,8-tetrachlorodibenzo-p-dioxin ("dioxin") that is expected to protect wildlife which use the Great Lakes Basin and ingest this chemical through their drinking water or food

sources. In developing this criterion, risks to specific wildlife species, including bald eagles, have been estimated.

5. I have been asked to focus this Declaration specifically on whether or not a long-term average ambient surface water concentration of dioxin of 0.013 parts per quadrillion ("ppq") can be reasonably assumed not to have an adverse effect on bald eagles that use a waterbody containing this concentration of dioxin as a site to obtain food and drinking water.

6. To determine if a chemical in a waterbody endangers fish or wildlife due to ingestion of the pollutant, assessments of how much of the chemical is consumed and how toxic the chemical is to the animals must be estimated. These two analyses are termed the exposure assessment and the hazard assessment, respectively. Taken together, these analyses are generally termed a risk assessment. For example, to estimate the amount of chemical a specific bird species consumes it is necessary know the amount of water they drink and how much contaminated food they eat. To assess toxicity of the chemical, a relationship between the dose of the chemical and the severity of toxic effects is required. From this dose-response curve a "no adverse effect level" can be estimated that represents a dose that can reasonably be assumed to cause no harmful effects to the species in question. Once the exposure and hazard assessments have been performed, it is possible to begin

making judgements about how protective an ambient surface water concentration would be for a specific avian wildlife population.

7. All chemical risk assessments, including the analysis which will be presented in this Declaration, incorporate assumptions because there exist uncertainties in the scientific understanding associated with the exposures to and hazards of pollutants. An example of one source of uncertainty lies in the extrapolation of results from direct toxicological tests on common laboratory animals, like the rat or chicken, to those that could be expected in humans or endangered wildlife species, which of course cannot typically be tested directly. Because of this and other uncertainties, it is not possible to state with absolute confidence that any given water concentration is completely "safe" to wildlife populations, but rather calculations can be performed that adjust for these uncertainties and provide estimates of chemical concentrations that can be reasonably assumed to have no adverse effects. When performing risk assessments it is also important to acknowledge when uncertainties associated with a specific analysis become so large that a reasonable scientific judgement cannot be made. As described in paragraph 8, I do not believe this to be the case for an assessment of dioxin risk to bald eagles. It must also be acknowledged in this specific instance that the technical basis for preparing assessments of chemical risks to wildlife is an evolving science within the U.S. EPA, as well as within other federal and state regulatory agencies. Consequently, refinements

to wildlife risk assessment methods over the next decade are expected.

8. In my professional opinion, an ambient surface water concentration of dioxin of 0.013 ppq can be reasonably assumed to cause no adverse effects to bald eagle populations exposed to the chemical through their drinking water and food consumption. The logic behind my opinion is outlined below and is based on the collaborative efforts mentioned in paragraph 4.

a. Bald eagles are exposed to dioxin in surface waters primarily by eating fish and by drinking from the surface waters. To perform a dioxin exposure assessment for bald eagles it is necessary to estimate the specific nature of their diet, the dioxin content of their diet, the typical weight of adult eagles, their dietary intake rates (i.e., kilograms of food consumed per day), their drinking water intake rate (i.e., liters of water consumed per day) and the dioxin content of the surface waters.

b. I and my colleagues in the Great Lakes Basin project reviewed the scientific literature in order to establish estimates for the bald eagle exposure parameters. To the best of my knowledge, the literature indicates that bald eagles consume a variety of food including fish, waterfowl and small mammals, both as live prey and carrion (as reviewed by Colborn, 1991). However, if available, fish are their principal food and may make up 100% of

their diet (Newell et al., 1987; Palmer, 1988; Kozie and Anderson, 1991). For purposes of this analysis it is assumed that a bald eagle's diet is entirely fish. Based on an analysis of the scientific literature it is my opinion that it can be reasonably assumed that an adult bald eagle weighs about 4.5 kilograms (Bortolotti, 1984; Stalmaster and Gessaman, 1984; Palmer, 1988) and consumes 0.50 kilograms of fish per day (Stalmaster and Gessaman, 1982; Stalmaster and Gessaman, 1984; Craig et al., 1984). Using an avian-specific allometric equation (i.e., an equation that relates water intake to the weight of an animal) of Calder and Braun (1983), the drinking rate of a 4.5 kilogram eagle can be estimated to be 0.16 liters per day.

c. For purposes of this risk assessment it will be assumed that all of the surface waters from which a bald eagle consumes fish and drinks water contains 0.013 ppq dioxin. Estimating dioxin exposure to bald eagles through drinking water is straightforward; however, estimating the dioxin content in fish, and thus dioxin exposure to bald eagles though their diet, is more complex. Dioxin is known to accumulate in fish tissues at concentrations greater than are present in the surrounding water. The degree of dioxin bioaccumulation in fish will vary with the percent fat content of the fish (i.e., the less fat, the lower the degree of bioaccumulation) and the amount of dissolved and suspended organic materials in the water (i.e., the more dissolved or suspended organic matter the lower the bioaccumulation). There is evidence

that indicates that dioxin bioaccumulation increases by a factor of 10,000 for every 1% increase in percent fat content of fish residing in a large-cold temperature waterbody with low levels of dissolved or suspended organic matter (Cook et al., 1991; Cook, 1992). If such fish contain 9% fat, it can be estimated that dioxin will accumulate in the fish to a level 90,000 times greater than that present in the surrounding water. This calculation is based on the assumption that fish-eating wildlife consume whole fish carcasses (i.e., they do not selectively consume fish fillets which have a lower fat content). The results of several studies performed in the Great Lakes Basin indicate whole fish carcasses have an average fat content of 7.9% (U.S. EPA, 1991). Although these studies suggest that for a large, cold-temperature waterbody the average percent fat content in fish was approximately 8%, it has been proposed that a value of 9% be used to protect for the variation in the measurements (U.S. EPA, 1991).

d. Using the assumptions outlined in points 8a, 8b, and 8c, the dioxin dose to adult bald eagles eating fish and drinking water from a waterbody containing 0.013 ppq dioxin can be estimated to be 130 picograms ("pg") of dioxin per kilogram eagle per day.

e. To assess the specific hazard of dioxin to eagles and other avian species, I and my colleagues in the Great Lakes Basin guidance project reviewed the scientific literature for toxicity studies with avian species that involved administration of dioxin

either orally or intraperitoneally[1] ("i.p.") and which generated sufficient data to establish a dose-response curve (see paragraph 6). Consistent with the other general requirements employed in the Great Lakes Basin project, suitable studies were also required to be of at least 28 days in length and include measurements of reproductive impairment. These requirements were used to insure that tests were long enough to find adverse effects other than death, including reproductive impairment. For the purposes of performing the dioxin hazard assessment, priority was given to those studies involving wildlife species. To the best of my knowledge, there are no studies on bald eagles that satisfy these requirements (presumably because they are protected by the Endangered Species Act). Further, to the best of my knowledge the only study that meets the test requirements is that of Nosek (1991), where effects of dioxin on the ring necked pheasant were investigated.

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[1]An intraperitoneal dose means the chemical is administered with a syringe and needle by injecting the compound through the membrane that lines the wall of the abdominal cavity and encloses the internal organs. Oral routes of dosing can include using a syringe and blunt needle to administer the chemical to the stomach or, more commonly, by either treating feed or water with the chemical and providing the contaminated food source or drinking water to the animals. Oral routes of exposure were considered most similar to the routes of exposure to be expected for wildlife using surface waters. It is also generally acknowledged in toxicology that i.p. and oral routes of exposure are similar because in both instances the chemical is absorbed first by important internal organs such as the liver. Given that dioxin is extremely hazardous to humans, and the potential for worker contamination is greater in a feeding or drinking experiment, it was determined that studies using an i.p. route of exposure would be accepted, since researchers would likely employ this method to protect themselves and their co-workers.

f. In the Nosek (1991) study pheasants were dosed i.p. once a week, for 10 weeks, at rates equivalent to 0, 1,400, 14,000 and 140,000 pg of dioxin per kilogram of pheasant per day. A significant decrease in both egg hatchability and the number of eggs produced per hen were observed in the group that was exposed to 140,000 pg of dioxin per kilogram pheasant per day. A significant increase in embryo mortality was also observed at that dose. However, no significant effects on egg hatchability, the number of eggs produced or embryo mortality were observed in the birds exposed to 0, 1,400 and 14,000 pg of dioxin per kilogram pheasant per day. Thus, the study demonstrated a no effect level of dioxin at 14,000 pg of dioxin per kilogram pheasant per day.

g. If it were assumed that bald eagles were equally as sensitive to dioxin as pheasants, the estimated intake of 130 pg dioxin per kilogram bald eagle per day associated with a water concentration of dioxin of 0.013 ppq (see point 8d) would be approximately one hundred times lower than an eagle no effect level of 14,000 pg dioxin per kilogram bald eagle per day.

h. In my professional opinion, it is reasonable to assume that bald eagles may be more sensitive than pheasants to dioxin exposure. Therefore, in my opinion, it would be appropriate to adjust the pheasant no observed adverse effect level to estimate the hazard of dioxin exposure to bald eagles. The use of data from surrogate species to estimate the response in another species is a

well established practice in toxicology. For example, adverse effects of chemicals on humans are typically estimated through use of laboratory studies involving rats, mice, and dogs. A ten-fold "uncertainty" factor is commonly employed (U.S. EPA, 1990) to extrapolate toxicological data from laboratory animals to humans (i.e., the no adverse effect dose for the human is estimated by dividing the no adverse effect dose obtained from the surrogate species by ten). To account for the possibility that the bald eagle may be more sensitive than the ring necked pheasant, the no effect level for pheasants of 14,000 pg dioxin per kilogram pheasant per day can be divided by 10 to give a more protective value of 1,400 pg dioxin per kilogram bald eagle per day.[2] Therefore the estimated intake of 130 pg dioxin per kilogram bald eagle per day associated with a water concentration of dioxin of 0.013 ppq (see point 8d) would be approximately ten times lower

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[2]Based on studies comparing the sensitivity of ring necked pheasants and chickens to polychlorinated biphenyl intoxication, the 10-fold uncertainty factor is, in my opinion, reasonable in this specific instance. The chicken is generally accepted as the most sensitive avian species with regard to intoxication by specific chemicals that are believed to have the same mode of toxic action as dioxin, such as certain polychlorinated biphenyls (e.g., see review by Gilbertson et al., 1991). An analysis of several well-conducted chicken and pheasant polychlorinated biphenyl studies (Dahlgren et al., 1972; Scott, 1977; Lillie et al., 1974; Britton and Huston, 1973) indicate that the chicken is approximately 3 fold more sensitive than the pheasant. The use of a 10-fold uncertainty factor in the dioxin assessment presented here assumes the eagle may be more sensitive than the chicken to intoxication by dioxin-like chemicals and is, in my opinion, appropriately conservative.

than an eagle no effect level of 1,400 pg dioxin per kilogram bald eagle per day.

i. Because there is variability in sensitivity within any species, an adjustment of the no effect level to provide protection of sensitive individuals within an endangered species, such as the bald eagle, would, in my opinion, be appropriate. To provide for protection of sensitive eagles, the bald eagle no effect level of 1,400 pg dioxin per kilogram bald eagle per day (see point 8h) can be divided by 10 to give an estimated no adverse effect value of 140 pg dioxin per kilogram bald eagle per day. The use of a 10-fold "uncertainty" factor to perform extrapolations of toxicological data within a species is typically employed in human hazard assessments for noncarcinogens to provide for an increased level of protection corresponding to protection of sensitive individuals (U.S. EPA, 1990). The estimated intake of 130 pg dioxin per kilogram bald eagle per day associated with a water concentration of dioxin of 0.013 ppq (see point 8d) is lower than an eagle no effect level of 140 pg dioxin per kilogram bald eagle per day.

j. Because the estimated dose of 130 pg dioxin per kilogram eagle per day, which is associated with a water concentration of dioxin of 0.013 ppq (see point 8d), is less than the estimated no adverse effect dose for bald eagles that is based on a series of uncertainty assumptions (see points 8f, 8g, 8h, and 8i), it is my

professional opinion that it is reasonable to assume that no adverse effects to bald eagles, which are attributable to dioxin ingestion, will result from the presence of dioxin in surface waters at a concentration of 0.013 ppq.

10. It is my professional opinion that it is scientifically reasonable at this time to assume that an ambient surface water concentration of dioxin of 0.013 ppq will not result in adverse effects to bald eagle populations exposed to the chemical through their drinking water and food sources. In my opinion, this analysis is based on an assessment of dioxin hazards to bald eagles that can be considered broadly applicable, and upon an exposure assessment I consider a reasonable default analysis in the absence of site-specific information. As discussed in paragraph 7, future research may reduce uncertainties associated with dioxin risk assessments and result in a more refined analysis.

11. Finally, I have reviewed the Declaration of Dr. Ian Nisbet, dated December 23, 1991, and would like to address two issues he presents in Paragraph 15.

a. In Paragraph 15 of his Declaration Dr. Nisbet cites an unpublished study of Garrett et al. (1988) for the propositions that bald eagle diets in the Columbia River basin are comprised of 71% fish and that they also consume fish eating birds (quantity not specified) so that bald eagles are exposed to higher levels of

hazardous substances than are birds which only eat fish. Dr. Nisbet also cites the study of Kozie and Anderson (1991) to further assert that fish eating birds comprise a portion of bald eagle diets (quantity not specified). Dr. Nisbet fails to acknowledge that the value of 71% is based on an analysis of prey remains and that this technique tends to underestimate fish consumption (Kozie and Anderson, 1991 and references cited therein). In fact, Garrett et al. (1988) acknowledge this fact and state that fish actually account for either 90% (p. 64 of Garrett et al.) or 94% (p.65 of Garrett et al.) of the diet of bald eagles in the lower Columbia River. This conclusion is also consistent with the findings of Kozie and Anderson (1991) who found that an analysis of prey remains underestimates fish consumption. Indeed Kozie and Anderson (1991) estimate that fish actually represent 97% of an eagle diet. Consistent with the analysis discussed in Paragraph 8b of my Declaration, and the data provided above, in my opinion, it is reasonable to assess the risks of dioxin to bald eagles using an assumption that their diet is entirely comprised of fish.

b. Dr. Nisbet also asserts in Paragraph 15 of his Declaration that there is a probability (which he does not quantify) that dioxin contamination at existing levels contributes to reproductive impairment in bald eagles inhabiting the Columbia River Basin. He also asserts that, "Continued release of 2,3,7,8-TCDD into the system would further augment exposure of the eagles ... and hence adverse effects, such as reproductive impairment, would also be

augmented." To the extent that Dr. Nisbet is arguing that any continued release of dioxin to the Columbia River Basin, presumably even at drastically reduced levels compared to historic discharges, could augment adverse effects of dioxin to eagles as a result of augmented dioxin exposure, I believe that his argument lacks a scientific foundation. Dr. Nisbet does not support his statement with a scientific calculation that relates dioxin release to eagle exposure. His Declaration implies that a reduction in the concentration of dioxin in water, and therefore a reduction of dioxin concentrations in food, would result in an increase in dioxin exposure to eagles. In my opinion, this assertion is not intuitive and a scientifically-based argument or model is needed to establish that a decrease in dioxin concentrations in drinking water and food sources would lead to an increase in dioxin exposure to bald eagles. In my opinion, the dioxin body burden in bald eagles will be reduced over time if dioxin levels in their food and drinking water is reduced (assuming all other routes of exposure remain constant).

I declare that the foregoing is true and correct to the best of my knowledge. Executed this 12th day of February, 1992, in Duluth, Minnesota.

Steven P. Bradbury

REFERENCES

Britton, W.M. and T.M. Huston. 1973. Influence of polychlorinated biphenyls in the laying hen. Poultry Sci. 52:1620-1624.

Bortolotti, G.R. 1984. Sexual size dimorphism and age-related size variation in bald eagles. J. Wildl. Manage. 48:72-81.

Calder III, W.A. and E.J. Braun. 1983. Scaling of osmotic regulation in mammals and birds. Am. J. Physiol. 244:601-606.

Colborn, T. 1991. Epidemiology of Great Lakes bald eagles. J. Toxicol. Environ. Health. 33:395-453.

Cook, P.M., D.W. Kuehl, M.K. Walker and R.E. Peterson. 1991. Bioaccumulation and toxicity of TCDD and related compounds in aquatic ecosystems. Banbury Report 35: Biological Basis for Risk Assessment of Dioxins and Related Compounds. Cold Harbor Spring Press, pp. 143-167.

Cook, P.M. 1992. Personal communication. U.S. EPA, Environmental Research Laboratory-Duluth, Duluth, MN.

Garret, M., R.G. Anthony, J.W. Watson, and K. McGarigal. 1988. Ecology of Bald Eagles on the Lower Columbia River. Unpublished report to U.S. Army Corps of Engineers

Craig, R.J., E.S. Mitchell and J.E. Mitchell. 1988. Time and energy budgets of bald eagles wintering along the Connecticut River. J. Field Ornithol. 59:22-32.

Dahlgren, R.B., R.L. Linder and C.W. Carlson. 1972. Polychlorinated biphenyls: Their effects on penned pheasants. Environ. Health Perspect. 1:89-101.

Gilbertson, M., T. Kubiak, J. Ludwig and G. Fox. 1991. Great Lakes embryo mortality, edema, and deformities syndrome (GLEMEDS) in colonial fish-eating birds: Similarity to chick-edema disease. J. Toxicol. Environ. Health. 33:455-520.

Kozie, K.D. and R.K. Anderson. 1991. Productivity, diet, and environmental contaminants in bald eagles nesting near the Wisconsin shoreline of Lake Superior. Arch. Environ. Contam. Toxicol. 20:41-48.

Lillie, R.J., H.C. Cecil, J. Bitman and G.F. Fries. 1974. Differences in response of caged white leghorn layers to various polychlorinated biphenyls (PCBs) in the diet. Poultry Sci. 53:726-732.

Newell, A.J., D.W. Johnson and L.K. Allen. 1987. Niagara River biota contamination project: Fish flesh criteria for piscivorous wildlife. Technical Report 87-3 of the Division of Fish and Wildlife, Bureau of Environmental Protection, New York State Department of Environmental Conservation, Albany, NY.

Nosek, J.A. 1991. The effects of exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in ring-necked pheasants. Ph.D. Dissertation. University of Wisconsin, Madison. Note: The chapters associated with this dissertation have been accepted for publication in J. Toxicol. Environ. Health.

Palmer, R.S. (Ed.). 1988. Handbook of North American Birds: Volume 4. Yale University Press, New Haven, CT.

Scott, M.L. 1977. Effects of PCBs, DDT, and mercury compounds in chickens and Japanese quail. Federation Proceedings 36(6):1888-1893.

Stalmaster, M.V. and J.A. Gessaman. 1982. Food consumption and energy requirements of captive bald eagles. J. Wildl. Manage. 46:646-654.

Stalmaster, M.V. and J.A. Gessaman. 1984. Ecological energetics and foraging behavior of overwintering bald eagles. Ecol. Monogr. 54:407-428.

U.S. EPA. 1990. General quantitative risk assessment guidelines for noncancer health effects. Draft. U.S. EPA, Office of Research and Development, Washington, DC.

U.S. EPA. 1991. Great Lakes water quality initiative technical support document for the procedure to determine bioaccumulation factors. Internal U.S. EPA draft document, November, 1991, U.S. EPA Region V, Chicago, IL.

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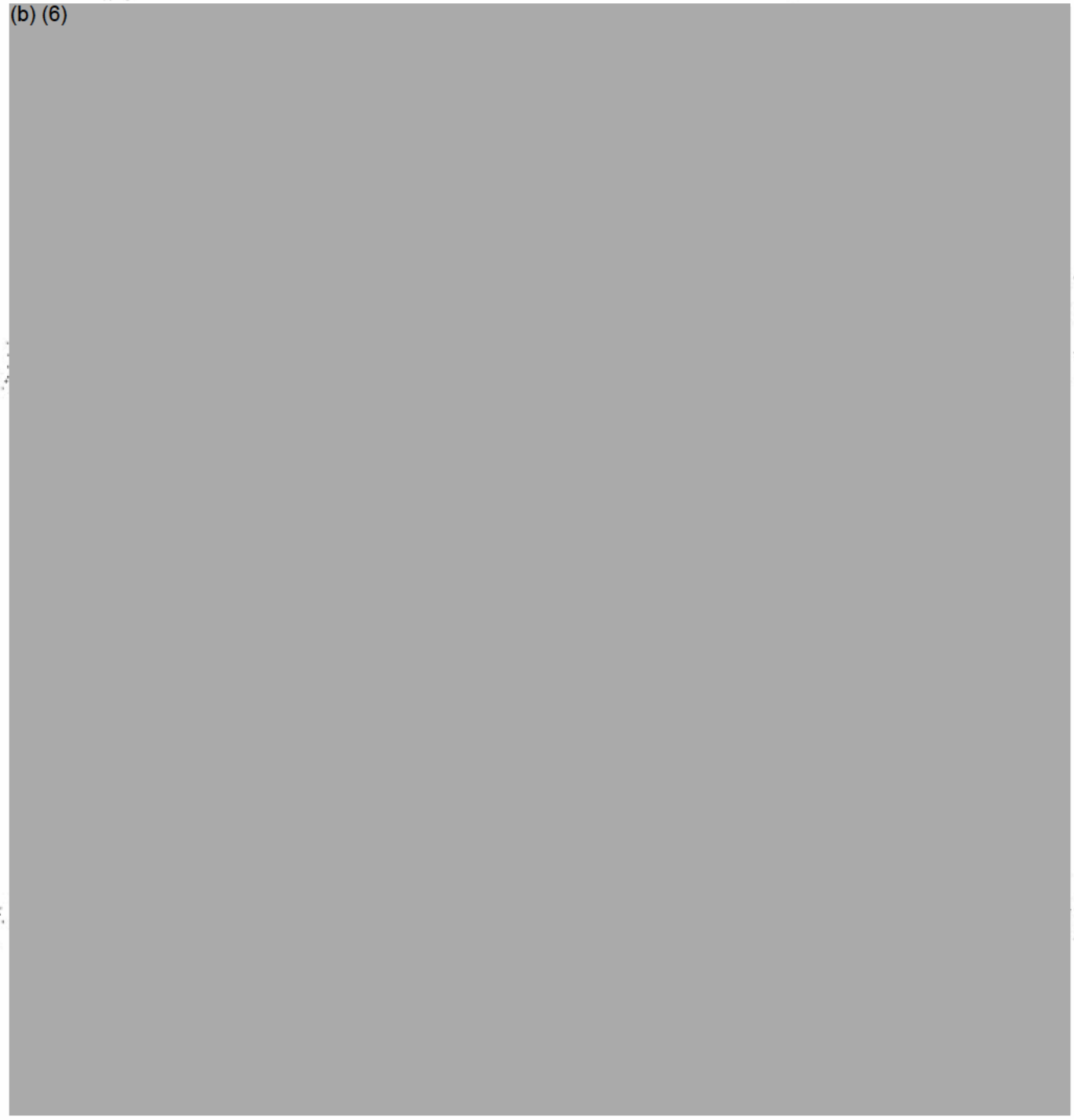

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